THE IMPACTS OF LEAD IN MICHIGAN

(A Science Report To Governor John Engler)

Prepared by
Michigan Environmental Science Board
Lead Panel

MICHIGAN ENVIRONMENTAL SCIENCE BOARD
LEWIS CASS BUILDING
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MARCH 1995

(FIRST PRINTING)

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1st Printing March, 1995: 1000 Copies $1,550.00
(Cost per Copy: $1.55)

Printed by Authority of Executive Order 1992-19

Correct Report Citation:

Bulkley, J.W., R.Y. Demers, D.T. Long, G.T. Wolff and K.G. Harrison. 1995. *The Impacts of Lead in Michigan*, March, 1995. Michigan Environmental Science Board, Lansing. xii + 42p.

PREFACE

Michigan Environmental Science Board

The Michigan Environmental Science Board (MESB) was created by Governor John Engler by Executive Order 1992-19 on August 6, 1992. The MESB is charged with advising the Governor, the Natural Resources Commission, the Michigan Department of Natural Resources and other state agencies, as directed by the Governor, on matters affecting the protection and management of Michigan's environment and natural resources. The MESB consists of 9 individuals and an executive director, appointed by the Governor, who have expertise in one or more of the following areas: engineering, ecological sciences, economics, chemistry, physics, biological sciences, human medicine, statistics, risk assessment, geology and other disciplines as necessary. Upon the request of the Governor to review a particular issue, a panel, consisting of MESB members with relevant expertise, is convened to evaluate and provide recommendations on the issue. The MESB is neither a state policy body nor an advocate for or against any particular environmental or public health concern.

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TABLE OF CONTENTS

LIST OF TABLES AND FIGURES

Page

The Impacts of Lead in Michigan

MAJOR FINDINGS AND CONCLUSIONS

On February 25, 1994, the Michigan Environmental Science Board was charged by Governor John Engler to identify and rank the various routes of human lead exposure; prioritize the most effective targets for remediation in terms of human exposure reduction, paying special attention to reducing exposure to children; and identify the efficacy of various remediation techniques for lead.

On March 21, 1994, a Lead Panel, composed of five MESB members, was convened to begin the project. The investigation consisted of the accumulation and evaluation of peer-reviewed and some non-peer-reviewed literature and data on the subject. In addition, verbal and written testimony from industry and health specialists, state regulatory agencies and concerned citizens was considered. Major findings and conclusions of the Panel are summarized below.

► Sources of lead are pervasive throughout society, and perhaps even more so in Michigan because of the industrial nature of the state. Lead exposure prevention is critical to Michigan, because lead poisoning is a preventable condition and because the insidious and subclinical outcomes of lead exposure may result in significant decreases in overall health and productivity of the state's citizens. Many lead poisoned children are asymptomatic. The impacts of elevated blood-lead levels in children may result in deficits in cognitive functions, neurophysiological difficulties and problems in school.

► The elimination of the use of lead-based gasoline and the reduced use of lead-based paints have measurably reduced the risk of elevated blood-lead levels to the general population. However, a calculated exposure level estimated to be the average currently in Michigan children roughly corresponds to the Centers for Disease Control's (CDC) 1991 designated blood-lead level of concern (10 µg/dL). As a consequence, there would appear to be little or no margin of safety for any additional incremental exposure in a household from peeling and deteriorating lead-based paint or lead-contaminated drinking water supply distribution systems. In addition, any incidence of pica would result in a blood-lead level in excess of the CDC's level of concern of 10 µg/dL.

► Within the scientific and medical communities there is consensus that the primary routes of lead exposure in children are ingestion of lead-based paints and lead-containing dusts and soil. In occupational settings, the primarily route in adults is inhalation of lead-containing dusts and fumes. The belief, that in order to be poisoned, children must eat lead paint chips, is unfounded. The most common cause of lead poisoning in children is ingestion through hand-to-mouth transmission of lead paint-contaminated surface dust.

► Screening is essential in order to identify children with elevated blood-lead levels. Accurate and complete data regarding exposure and blood-lead levels in populations at risk are essential in order to design and implement an effective intervention and control strategy. However, comprehensive and reliable blood-lead data are not presently available for either Michigan children or adults. Limited blood-lead data available on lead

exposure among Michigan children from three pilot study areas (city of Detroit; Ingham, Kent, Muskegon and Saginaw Counties; and Wayne County) appear to be under-reported, fragmented and incomplete. This may be due in part to the fact that the three Michigan pilot programs are responsible to different federal funding agencies with different reporting requirements or expectations. In addition, the collection, reporting and management of these data among the pilot areas are not uniform. The state's adult lead program, partly because of the lack of promulgated rules and mandatory reporting, has an even more serious data collection problem.

► There is a need for the state to: (1) uniformly establish the use of a venous/direct blood-lead sampling/testing protocol, and to uniformly use 10 µg/dL as the blood-lead level reporting statistic for children across the state; (2) assume a coordinating role of all federally-funded lead exposure, prevention and abatement projects in Michigan; (3) establish a statewide, uniform and coordinated data collection, reporting and management system to capture and accurately portray the incidence of elevated blood-lead levels in both children and adults; (4) establish a lead registry for both children and adults; and (5) promulgate rules which will help enforce reporting of elevated blood-lead levels for both children and adults.

► Based on the literature and the limited blood-lead levels and demographic data for the state, the largest population group and also the one most at risk for elevated blood-lead levels within Michigan would be lower socio-economic urban preschool children (< 6 years old) living in older (pre 1980's) homes.

► The state should further investigate and consider the piloting of a community-based urban lead hazard reduction program similar to that which is currently operating in Baltimore, Maryland in order to reduce lead exposure in urban Michigan preschool children. The program should include the following components: (1) Development of a comprehensive lead-exposure data base, (2) Identification and prioritization of lead-based paint problem areas, (3) Abatement of the critical lead-based paint problems and (4) Education of the resident families on home maintenance and nutrition.

► While representative of a potential future hazard, intact and well-maintained lead-based paint should not be abated. Lead-based painted surfaces become a hazard only when they have been allowed to deteriorate. Lead-contaminated dust is generated as lead-based paint deteriorates over time, is damaged by moisture, abraded on friction and impact surfaces, or disturbed in the course of renovation, repair or abatement projects. One of the main reasons, other than lack of maintenance, for lead-based paint deterioration is trapped moisture. As a consequence of most homes becoming energy efficient, moisture is more easily trapped and undermines most painted surfaces. Homeowners with currently intact lead-based paint and contractors who abate homes with deteriorating lead-based paint should be made aware of the moisture problem and the methods to correct it.

► Inadequate studies preclude any valid determination as to which of the specific in-place management and abatement techniques is the most effective. Future, well designed and controlled paint, soil, and dust lead-abatement studies are needed which

will incorporate blood-lead measurements. However, based on the available lead-based paint, dust and soil hazard control techniques data, and on the growing evidence linking exposure to lead-containing dust to elevated blood-lead levels in children, the most efficacious residential lead-based paint abatement protocol should include those methods which employ the least invasive, least dust generating in-place management and abatement technique(s) available.

► Regardless of the residential lead-based paint remediation methodology employed, all should be followed with education for the adult occupants regarding identification and management of lead-based paint hazards, periodic and proper house cleaning and maintenance procedures, and proper nutrition and hygiene for children living in the home. This may be of particular importance for children with blood-lead levels below 30 µg/dL given the possible negative impact of current lead abatement techniques on lower blood-lead levels. Additional controlled studies are also needed to better evaluate the overall impacts of both education and sound nutrition on lead-abatement effectiveness.

THE IMPACTS OF LEAD IN MICHIGAN

(A Science Report to Governor John Engler)

INTRODUCTION

The Michigan Environmental Science Board (MESB) was created by Governor John Engler by Executive Order 1992-19 on August 6, 1992. The MESB is charged with advising the Governor, the Natural Resources Commission, the Michigan Department of Natural Resources and other state agencies, as directed by the Governor, on matters affecting the protection and management of Michigan's environment and natural resources. The MESB consists of an executive director and nine individuals appointed by the Governor. Each member has expertise in one or more of the following areas: engineering, ecological sciences, economics, chemistry, physics, toxicology and biological sciences, human medicine, statistics, risk assessment, geology and other disciplines as necessary. Upon the request of the Governor to review a particular issue, a panel, consisting of MESB members with relevant expertise, is convened to evaluate and provide recommendations on the issue.

On February 25, 1994, the MESB was charged by Governor Engler (Engler, 1994) to evaluate the concerns about lead in Michigan in order to provide guidance to state policy-makers (see Appendix I). On March 21, 1994, a Lead Panel, composed of five MESB members, was convened to begin the investigation. The investigation consisted of the accumulation and evaluation of peer-reviewed and some non-peer-reviewed literature and data on the subject. In addition, verbal and written testimony from industry and health specialists, state regulatory agencies and concerned citizens was considered at eight meetings (Harrison, 1994a; 1994b; 1994c; 1994d; 1994e; 1994f; 1994g; 1994h). The report was prepared by the Panel members with each individual assigned a specific topic or topics to address. The investigation lasted for a period of 11 months.

The report addresses three specific directives from the Governor:

1. Identify and rank the various routes of lead exposure in terms of level of human exposure;

2. Prioritize the most effective targets for remediation in terms of human exposure reduction, paying special attention to reducing exposure to children; and

3. Identify the efficacy of various remediation techniques for lead.

DIRECTIVE 1. Identify and rank the various routes of lead exposure in terms of level of human exposure.

Introduction

Lead as an element is present in the land, water, and air. The adverse effects of lead on human health have been documented throughout the ages (Lippman, 1990; Needleman, 1980). The use of lead in human societies has evolved over time. As civilizations and societies developed, lead played a role in a host of common products and activities and, as a consequence, became widely dispersed. According to Nriagu (1994), the body burden of lead in ancient peoples has been estimated to be 1/300th of the body burden of lead in humans today. Based on evidence from lead analysis of tree rings, polar ice caps and human bones, society's use of lead, especially since the industrial revolution, has increased human exposure to approximately 1,000 to 5,000 times higher than naturally occurring levels (Pounds, 1994).

Efforts to reduce global lead exposure, such as the removal of lead in gasoline, have resulted in decreased blood-lead levels. In the United States (U.S.), national lead emissions estimated for the years 1976 to 1992 show a decrease from 153,100 metric tons/year in 1976 to 5,200 metric tons/year in 1992 (USEPA, 1993; 1986b). The removal of lead from both gasoline and paint have been major factors contributing to this observed reduction of lead emissions (Clements International Corporation, 1993; CDC, 1991).

According to Pounds (1994), Clements International Corporation (1993), Centers for Disease Control and Prevention (CDC, 1991), and Agency for Toxic Substances and Disease Registry (ATSDR, 1988), elevated blood-lead levels greater than 10 µg/dL may adversely impact the central nervous system, peripheral nervous system, reproductive system, endocrine system, cardiovascular system, pulmonary system, gastric intestinal tract, and skeleton (bone is a reservoir for excess lead). Elevated blood-lead levels may result in behavioral disorders, perceptual motion deficits, mental retardation, seizures, and ultimately, death (Pounds, 1994).

For children in the age group six months to six years, the diagnosis of elevated blood-lead levels (e.g., **≥**10 µg/dL) is difficult without blood screening because most children are asymptomatic (Bhambhani, 1994). However, many of the results of elevated blood-lead may be significant, permanent and not fully manifested until later in life. According to Pounds (1994), the adverse and persistent effects may include decreased intelligence, reduced short-term memory, reading disabilities, deficits in vocabulary, deficits in fine motor skills, deficits in reaction time, deficits in hand-eye coordination, and in one study (Needleman *et al*., 1990), a seven-fold increase in failure to graduate from high school. According to the National Center for Education in Maternal and Child Health (NCEMCH, 1994) and the CDC (1991), the illness and adverse health impacts resulting from elevated blood-lead levels are preventable.

The purpose of this portion of the report is to identify and define the extent and impacts of lead exposure on Michigan children and adults.

Populations at Risk

Within the literature, there is a general consensus (National Center for Lead-Safe Housing, 1994; NCEMCH, 1994; Clements International Corporation, 1993; ATSDR, 1988), that the two populations most at risk for lead poisoning within the U.S. are urban preschool-aged children (< 6 years old) and adults with unprotected occupational exposures. Similar conclusions have been suggested for Michigan (Bhambhani, 1994; Pounds, 1994; Chiodo, 1994; and Rosenman, 1994).

Incidence of Lead Poisoning in Michigan Children. Comprehensive blood-lead data at the level of concern (10 µg/dL) considered by the CDC (1991) to represent excessive lead exposure in children are not presently available for Michigan. Limited data are available, however, for Michigan children at or above the 15 µg/dL blood-lead level. Table 1 presents more recent data for Michigan children (< 6 years old) from three Michigan urban pilot "Lead Poisoning Prevention and Control Programs" currently underway in (1) city of Detroit (DHD, 1994), (2) Ingham, Kent, Muskegon, and Saginaw Counties (MDPH, 1994a; VandenBosch, 1994), and (3) Wayne County (minus the city of Detroit) (WCDPH, 1994). During the period July 1, 1993 to June 30, 1994, a total 33,668 children (< 6 years old) were screened for elevated blood-lead levels within the three pilot areas. Of these, 1,745 children were confirmed (venous blood samples) with blood-lead levels greater than or equal to 15 µg/dL. An extrapolation of the observed relationship of confirmed to screened children, to the total number of children (< 6 years old) within the combined three pilot project area (364,020; 1990 U.S. Census Data) suggests that a total of 18,856 children or 5.18% of the total population would be expected to have elevated blood-lead levels equal to or greater than 15 µg/dL.

Table 1. Racial/ethnic origin of Michigan children (< 6 years old) screened and confirmed with blood-lead levels ≥15 µg/dL, 1993-1994. **(1)**

Category	Black	White	Hispanic	Asian/ Pacific Islander	Amer. Indian/ Alaska Native	Unknown (a)	Total
Children Screened (% Total Screened)	17,893 (53.2)	8,324 (24.7)	1,417 (4.2)	417 (1.2)	368 (1.1)	5,249 (15.6)	33,668
Children Confirmed (% Total Confirmed)	840 (48.1)	381 (21.8)	38 (2.2)	7 (0.4)	32 (1.8)	447 (25.6)	1,745
% Confirmed of Children Screened by Racial/Ethnic Origin	4.69	4.57	2.68	1.67	8.69	8.52	5.18

(1) Data compiled from 1993/1994 Quarterly and Annual Lead Poisoning Prevention and Control Program reports for the city of Detroit and Ingham, Kent, Muskegon, Saginaw and Wayne Counties.
(a) Racial/ethnic origin not determined.

Limited racial/ethnic data are also available for children with confirmed elevated blood-lead levels of 15 µg/dL or greater (Table 1). In terms of recorded racial/ethnic origin, Black children comprised the largest percentage of the total children screened and confirmed (53.2% and 48.1%, respectively), followed by White (24.7% and 21.8%), Hispanic (4.2% and 2.2%), Asian/Pacific Islanders (1.2% and 0.4%), and American

Indian/Alaskan Native (1.1% and 1.8%). Racial/ethnic origin was not designated for 5,249 of the screened (15.6%) and confirmed (25.6%) children. Table 1 also presents the percentage of confirmed to screened children within each racial/ethnic grouping. In relative terms, these observed data suggest that within racial/ethnic groups, American Indian/Alaskan Native children may be at higher risk (8.69%) of having elevated blood-lead levels than Black (4.69%), White (4.57%), Hispanic (2.68%) or Asian/Pacific Islander (1.67%) children. Given the limited nature of these data and the large undefined segment of the population (5,249 children of unknown racial/ethnic origin), it is not possible to reach any firm generalization on this topic. It may be necessary to increase future screening efforts for the extreme groups to see if the observed relationship holds true.

A word of caution must be expressed regarding the results obtained in Table 1. The data obtained from the three pilot projects contain several inherent limitations. First, the racial/ethnic data are based on a target blood-lead level of 15 µg/dL level rather than the recommended CDC (1991) value of 10 µg/dL due to the lack of such information at the 10 µg/dL level. As a consequence, all the calculated populations at risk in Table 1 under estimate the actual populations at risk at the CDC defined criteria of 10 µg/dL. Second and confounding the first problem further is the interspersed inclusion of blood-lead data obtained through the use of the capillary sampling and EP testing procedures rather than through the use of the more preferred and accurate (CDC, 1991) venous blood-lead sampling and direct-lead testing protocol. According to Scott (1994), the Michigan Department of Public Health (MDPH) laboratory ceased using the EP test in lieu of the direct lead test in June 1993; however, capillary sampling is still routinely used for lead screening purposes. When combined with additional MESB Lead Panel concerns, including an inability of the three pilot projects to differentiate a large segment of the sample population based on racial/ethnic background, errors in data calculations, and lack of follow up to verify or explain questionable data entries, these problems cast doubt on the overall reliability of the blood-lead data base for the three Michigan pilot projects to accurately reflect the true number of at-risk children by race or ethnic background.

The ATSDR (1988) has estimated the number of children (6 months to 5 years) who would be expected to have elevated blood-lead levels greater than 10 µg/dL and 15 µg/dL for 12 selected standard metropolitan statistical areas (SMSAs) within Michigan (Table 2). At the 10 µg/dL level, the estimates range from a high of 186,768 (56.5%) children in the Detroit SMSA to a low of 3,108 (30.5%) children in the Bay City SMSA. The total number of children of this age group expected to have an elevated blood-lead level greater than 10 µg/dL for all 12 SMSAs is 299,557 or 50.2% of the total child population. Similarly, the total number of children expected to have an elevated blood-lead level greater than 15 µg/dL is 114,076 (19.1%).

The ATSDR (1988) estimates in Table 2 are based on blood sample data collected in the late 1970's and therefore the child population projections of 50.2% (> 10 µg/dL) and 19.1% (> 15 µg/dL) may be overestimated due to the sampling and analysis techniques employed at that time (capillary sampling and erythrocyte protoporphyrin [EP] testing), changes in environmental and dietary lead and secular trends over time. The 1993/1994 pilot project projection (Table 1), although based for the most part on a more accurate venous blood sampling and direct lead testing protocol, is also limited by the number of

participating localities in the pilot projects and the number of screened children, suggesting that its calculated projection of 5.18% (≥15 µg/dL) may be an underestimation. While both sets of projections are based on reasonable assumptions, neither can be considered definitive. In addition, neither projection can be considered as reflective of the state as a whole.

Table 2. Estimated number of children (6 months to 5 years) for selected SMSAs in Michigan who are projected to exceed blood-lead levels of 10 µg/dL and 15 µg/dL. [(1)]

SMSA [(a)]	POPULATION (0.5yr-5.0yr)	POPULATION >10 µg/dL (>15 µg/dL)	PERCENTAGE >10 µg/dL (>15µg/dL)
Detroit	330,694	186,768 (77,492)	56. 5 (23.4)
Benton Harbor	14,393	6,690 (2,634)	46.5 (18.3)
Battle Creek	14,682	7,432 (2,549)	50.6 (17.4)
Flint	39,017	19,212 (6,707)	49.2 (17.2)
Muskegon	14,451	6,440 (2,375)	44.6 (16.4)
Saginaw	19,585	9,126 (3,092)	46.6 (15.8)
Jackson	11,694	5,201 (1,609)	44.5 (13.8)
Kalamazoo-Portage	22,318	9,144 (2,854)	41.0 (12.8)
Grand Rapids	58,978	22,941 (7,426)	38.9 (12.6)
Lansing-E.Lansing	41,420	16,515 (4,595)	39.9 (11.1)
Ann Arbor	19,647	6,980 (1,925)	35.5 (9.8)
Bay City	10,201	3,108 (818)	30.5 (8.0)
TOTAL	**597,080**	**299,557 (114,076)**	**50.2 (19.1)**

(1) Modified from ATSDR, 1988.
(a) SMSA = Standard Metropolitan Statistical Area.

Incidence of Lead Poisoning in Michigan Adults. Comprehensive data on adult occupational lead poisoning are also not available from the MDPH for the state. According to data from the Toxic Chemical Release Inventory (TRI88, 1990), there are 70 facilities within Michigan which reported the use of lead in manufacturing. Rosenman (1994) contends that these data represent an under-reporting by the lead-using facilities and suggests that the correct number of facilities should be closer to 1,400, based on an analysis of Michigan industries by Standard Industrial Classification (see Table 3).

Table 4 presents the types of occupations most likely to involve exposure to lead. Due to variations in state laws, it is difficult to obtain accurate data on the extent of elevated blood-lead levels resulting from exposure to these sources (ATSDR, 1990). Occupational exposures resulting in 50 µg/dL or greater must be reported to the state and federal Occupational Safety and Health Administrations; however, Michigan does not enforce the reporting requirement (Rosenman, 1994). According to Kalinowski (1994), MDPH receives a limited number of occupational disease reports from employers, physicians, hospitals, laboratories and clinics pertaining to elevated blood-lead levels. In 1993, Michigan only reported 88 cases (in 102 reports) with elevated blood-lead levels. Of the 88 individuals reported, 55 came from 12 different companies. The remaining 33 cases were either not related to occupational exposure or their employers were unknown. Fifty of the reports came directly from employers, 28 from laboratories and ten from private physicians (Rosenman, 1994). Rosenman (1994) contends that a more reasonable number of reports of adult occupational blood-lead poisoning for Michigan, given its size

and industrial base, should be between 800 and 1,500 cases per year.

Table 3. High risk industries for lead exposure in Michigan. **(1)**

SIC Code [(a)]	Type of Industry	Number of Facilities
3362	Brass, Bronze, and Copper Foundries	26
3341	Secondary Nonferrous Metal	10
3361	Aluminum Foundries	48
3479	Metal Coating and Allied Service	100
3494	Valves and Pipe fittings	48
3441	Fabricated Structural Metal	70
7538	General Automotive Repair Shops	80
3312	Blast Furnaces and Steel Mills	33
3369	Nonferrous Foundries	56
3471	Plating and Polishing	154
2851	Paints and Allied Products	36
5093	Scrap and Waste Materials	73
3443	Fabricated Plate Work (Boiler Shops)	51
3496	Misc. Fabricated Wire Products	47
3711	Motor Vehicles and Car Bodies	109
7539	Automotive Repair Shops	48
3231	Products of Purchased Glass	16
3316	Cold Finishing of Steel Shapes	17
3499	Fabricated Metal Products	67
3714	Motor Vehicle Parts and Accessories	315
TOTAL		**1,404**

(1) From Rosenman, 1994.
(a) SIC = Standard Industrial Classification.

According to Brown (1994) and VandenBosch (1994), enforcement of mandatory reporting of elevated blood-lead levels has been difficult in the absence of promulgated rules. As part of the rules process, the MDPH has developed a standardized blood-lead reporting form to be used by health providers and laboratories. The tenth revision of this form was reviewed by the MESB Lead Panel. Based on its review of the draft reporting form, the Panel expressed several concerns about its inadequacy, including, for example, its lack of data items requesting worker's occupation and employer. In an effort to help expedite the process, the MESB Lead Panel obtained and provided MDPH, for its consideration, copies of the blood-lead level reporting forms currently in use in California, Massachusetts, New Jersey, New York, and Ohio. Many of the items recommended by the MESB Panel were subsequently incorporated into the draft form. The proposed administrative rules and revised reporting form have since been published in the December, 1994 issue of the *Michigan Register* (MDPH, 1994b) beginning the formal rules promulgation progress.

Health Consequences of Excessive Human Lead Exposure

The purpose of this section is to summarize the current knowledge about the human health effects of lead exposure as they specifically correlate with blood-lead levels. Blood-lead is considered the principle biomarker for lead exposure since this test is widely used, reasonably easy to perform, low cost, and more reproducible and sensitive than other more indirect measures of lead exposure.

Lead exposure variably affects different populations. For example, risks are different for adults and children, and vary substantially by socioeconomic status. Adults are typically exposed by routes of inhalation, most often through their occupations, while children are most commonly exposed in the home environment by ingestion of lead-containing paint, dust and soil. Not only do adults and children differ by principle routes of exposure, but also by the fact that children tend to absorb a higher percentage of ingested lead than do adults (Behrman *et al*., 1992). Children absorb approximately 50% of ingested lead, compared to between 10% and 20% for adults. Further, children from impoverished backgrounds are more susceptible to the toxic effects of lead (Dietrich, Berger and Succop, 1993). Malnourishment is associated with a higher proportion of lead absorbed from the gastrointestinal tract. Also, nutritional anemia tends to increase the adverse effects of lead.

Table 4. List of lead exposure occupations. (1)

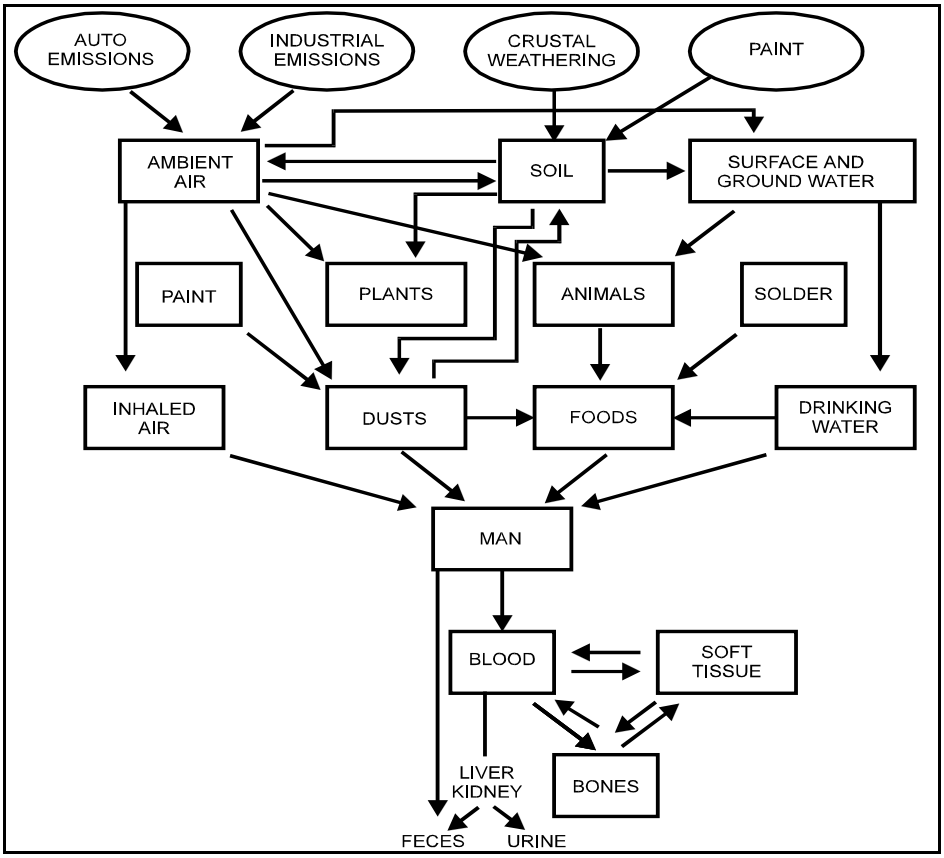
Plumbers, pipe fitters
Lead miners
Auto repairers
Glass manufacturers
Shipbuilders
Printers
Plastic manufacturers
Lead smelters and refiners
Police officers
Steel welders or cutters
Construction workers
Rubber product manufacturers
Gas station attendants
Battery manufacturers
Bridge construction workers

(1) Modified from ATSDR, 1990.

Routes of Exposure. Routes of exposure are summarized in Figure 1 (USEPA, 1986a). From the initial origins of automobile emissions, industrial emissions, crustal weathering and paint, numerous transfers of environmental sources ultimately result in lead being contained in the inhaled air, environmental dust, food, and drinking water. The main routes of entry into humans are inhalation and ingestion. Skin absorption is not a significant exposure route for lead. It is important to note the recent trend of decreasing lead usage in the U.S., and a corresponding decrease in total exposure. Total tonnage of lead use in the U.S. decreased from 206,000 tons in 1976 to 56,000 tons in 1983. The decrease in environmental levels of lead is highly correlated with diminished use of leaded gasoline (CDC, 1991; USEPA, 1986b). From 1976 to 1990, the lead content of gasoline has dropped by 99.8% (USEPA, 1991a). This decreased environmental load of lead correlates well with decreased amounts of lead levels contained in humans (Pirkle *et*

al., 1994). Figure 1 also illustrates the difficulty of controlling human exposure due to the multiple, inter-related pathways.

Figure 1. Pathways of lead from the environment to and within humans. **(1)**



(1) From USEPA, 1986a.

Exposure sources of principle concern in Michigan include lead-based paint, dust, soil, water, and gasoline. Paint is widely accepted as the principle source of exposure for children (Clements International Corporation, 1993; MTC, 1993). The route of exposure for children typically is ingestion, either of paint chips, or of dust emanating from deteriorating painted surfaces. Children aged one to six years are primarily at risk since ingestion comes from frequent hand-to-mouth contact in children of these ages. Drinking water can also serve as a source of exposure to humans, through leaching from lead-containing pipes and fixtures. Most investigators agree that lead-containing water results in only rare cases of human toxicity (Houk, Ing and Falk, 1989).

Lead-containing soil and dust can also contribute to human toxicity (Mielke *et al*., 1983). This is a particularly significant exposure source for those living in proximity to industries involved in the manufacture of lead products. For example, homes close to lead smelters may contain elevated amounts of lead in the soil, resulting in a potential exposure, especially to children (National Center for Lead-Safe Housing, 1994; ATSDR, 1988). Other exposures include various medicines, folk remedies, lead-containing ceramics, etc.

(MMWR, 1993). Such exposure sources can often be unpredictable, and unnoticed by clinicians and public health officials.

When considering sources of lead exposure, it is important to consider the total environmental burden in parallel with specific exposure to at-risk populations. Assessment of human lead exposure must take into account cumulative exposures from all sources. Body burden of total lead in humans is often the result of more than one source of exposure. For example, although a child's principle source of lead may have been ingestion of paint chips and dust inside of the home, outside soil and dust, ambient air lead levels, and contaminated drinking water may all have contributed to the total internalized dose (see Table 5).

Clinical Presentation. The diversity of presentation for individuals exposed to lead ranges from the clinically evident, to those which are much more subtle and subclinical. The subclinical presentations, though diagnostically difficult, may often result in permanent, long-term impairment. This spectrum of disease is reflected in the following examples (Landrigan, 1989).

The hemoglobin molecule is known to be altered at blood-lead levels of 10 µg/dL to 20 µg/dL, whereas anemia which is easily diagnosed does not develop until blood-lead levels have reached 40 µg/dL to 50 µg/dL. Nerve conduction is slowed in peripheral nerves at levels of 30 µg/dL, whereas obvious peripheral neuropathy, such as wrist drop, requires blood-lead levels of 60 µg/dL to 80 µg/dL. Lowered intelligence and behavioral change are noted, through neuropsychological testing of children, at levels of 10 µg/dL to 20 µg/dL, whereas acute encephalopathy requires blood-lead levels of 100 µg/dL or greater. Function of the proximal renal tubule is altered at levels of 25 µg/dL whereas renal failure is seen with blood-lead levels of 100 µg/dL. Current technologies provide for implementation of early clinical and public health intervention before more serious disease develops (Pounds, 1989).

Numerous organ systems are adversely affected by lead exposure (Pounds, 1994; Lockitch, 1993; Clements International Corporation, 1993; Schwartz *et al*., 1990). Neurological effects have long been known to result from both chronic and acute lead exposure. Encephalopathy, resulting in seizures, coma and death, is known to result from acute exposure and, as noted above, is correlated with blood-lead levels of 100 µg/dL to 140 µg/dL. Chronic, low grade exposure to lead can alter functional development of the brain, particularly during its vulnerable phase of growth in young children. Additionally, blood-lead levels even in the range of 10 µg/dL to 20 µg/dL can permanently alter central nervous system function, resulting in lowered intelligence, behavioral problems and poor school performance.

Both female and male-mediated reproductive toxicity is associated with lead toxicity. Female-mediated reproductive effects of high level lead exposure include increased rates of stillbirths, abortion, and neonatal death. A lowering of birth weight and gestational age are correlated with elevated maternal blood-lead levels. A significant association between maternal blood-lead and congenital malformation has not been consistently found in studies. Sperm counts in males are diminished at blood-lead levels of 30 µg/dL

to 50 µg/dL.

Table 5. Contributions from various media to blood-lead levels (µg/dL) of U.S. children (age = 2 years): background levels and incremental contributions from air. [(1)]

Source	Air Lead ug/m^3)						
	0	0.25	0.50	0.75	1.00	1.25	1.50
Background -- Non-air							
Food, water, and beverages	2.37	2.37	2.37	2.37	2.37	2.37	2.37
Dust	0.30	0.30	0.30	0.30	0.30	0.30	0.30
Subtotal	2.67	2.67	2.67	2.67	2.67	2.67	2.67
Background -- Air							
Food, water, and beverages	1.65	1.65	1.65	1.65	1.65	1.65	1.65
Ingested dust (with lead deposited from air)	0.00	1.57	3.09	4.70	6.27	7.84	9.40
Inhaled air	0.00	0.50	1.00	1.50	2.00	2.50	3.00
Total	4.3	6.4	8.4	10.5	12.6	14.7	16.7

(1) Modified from Lippmann, 1990.

Cardiovascular affects described in the recent literature are usually focused on elevations of blood pressure. Mild hypertension has been associated with a wide range of elevated blood-leads, varying between 10 µg/dL and 80 µg/dL, suggesting that there is no specific threshold for this effect. Several mortality studies (Michaels, Zoloth and Stern, 1991; Fanning, 1988; Malcolm and Barnett, 1982) have shown a positive association, though usually statistically insignificant, with cerebral vascular accidents as a cause of death among lead-exposed individuals. This effect may be the indirect result of elevations in blood pressure.

Kidneys are adversely affected by both acute and chronic exposures to lead. Heavy and acute lead exposure results in proximal tubular cell damage with aminoaciduria and glycosuria. More chronic exposures can cause renal tubular atrophy and interstitial nephritis, resulting in chronic renal failure. Risk of renal disease, in occupational exposures, has been associated with blood-lead levels of 40 µg/dL and 60 µg/dL.

Bone is a major target organ for lead. Skeletal lead has been used as a dosimeter of cumulative lead exposure (Pounds, 1994; Pounds, Long and Rosen, 1991). Lead is also stored in bone and accounts for approximately 90% of the body burden with chronic lead exposure. Lead can adversely affect bone resulting in impaired growth, delayed tooth eruption, alteration of vitamin D function, and reduced stature. In adults, possible associations of lead exposure to Paget's disease, and osteoporosis are being evaluated.

Hematologic effects are most visibly apparent with the onset of anemia. Anemia commonly results with blood-lead levels of 40 µg/dL or greater. In lead poisoning, erythrocyte protoporphyrin decreases and zinc protoporphyrin increases. Blood protoporphyrin has been used as a surrogate for lead exposure. However, a corresponding blood-lead level of at least 25 µg/dL to 35 µg/dL is required to show an elevated erythrocyte protoporphyrin level. Therefore, clinically significant blood-lead elevations of 10 µg/dL to 35 µg/dL may be missed. Lead-screening programs, especially for children, presently utilize blood-lead as the screening test of choice.

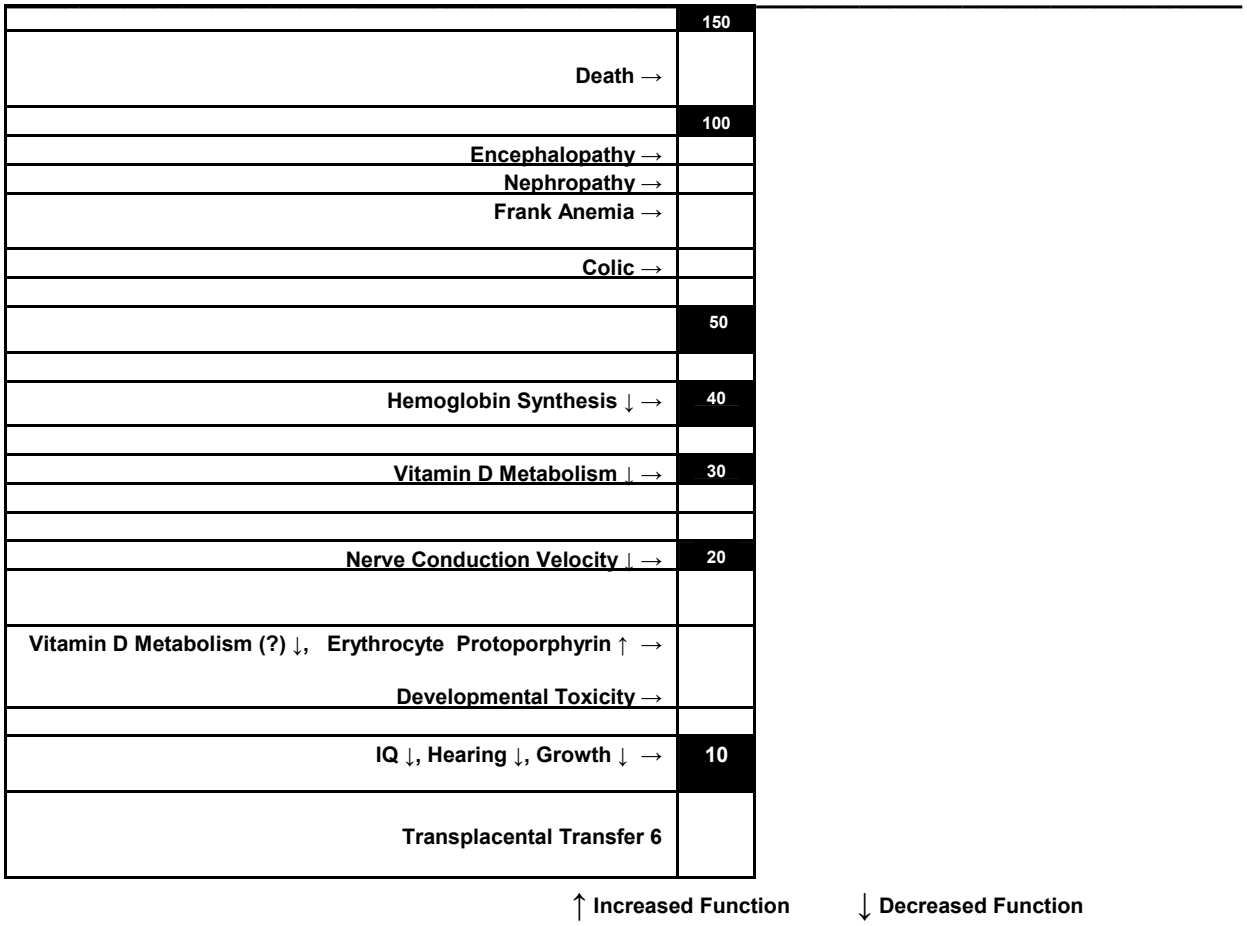
Special Effects in Children

A summary of the Lowest Observed Effect Levels of inorganic lead in children is shown in Figure 2 (CDC, 1991). Lead exposure among children warrants special concern, both because of their particular susceptibility, and because of the non-specific nature of early symptoms which may foretell significant long-term neurological outcomes (Chao and Kikano, 1993). Also, lead exposure among children, due to the fact that it is primarily from the source of household paint, is preventable. Other contributing sources of lead exposure, however, must not be forgotten. Drinking water has already been identified as one potential source. Also, exposure through parental occupation must always be considered. According to the CDC (1991), children of parents who are occupationally exposed to lead have higher blood levels of lead than their non-exposed counterparts.

The awareness of paint as the primary source of lead exposure in children has evolved over the recent decades. Screening of children in major U.S. cities in the early 1960's revealed that 20% to 45% of screened children had elevated blood-lead levels at 40 µg/dL (Cosgrove *et al*., 1989). These findings resulted in the Lead-Based Paint Poisoning Prevention Act passed by Congress in 1971. Societal awareness and legislation have since resulted in a steady lowering of childhood blood-lead levels. For example, in 1976, the percentage of children with blood-lead levels greater than 10 µg/dL was 78%, and this decreased to 4% in 1991 (Cosgrove *et al*., 1989).

By far the greatest concern of lead exposure among children today is related to its neurobehavioral effects. Most significant is the effect on mental development, as reflected in decrements of Intelligence Quotient (IQ) correlated with lead exposure (Dietrich *et al*., 1993). Blood-lead levels of 20 µg/dL or greater, compared to those of less than 10 µg/dL revealed an IQ difference of seven points for children averaging six and one half years of age at the time of the study, controlling for parental IQ. Numerous studies of the neurobehavioral effects of lead exposure have ensued in recent years, and have shown abnormalities in a number of outcomes related to nervous system development (Dietrich *et al*., 1993; Wasserman *et al*., 1992; Needleman and Gatsonis, 1990). A notable finding by Needleman *et al*., (1990) was the diminished school performance among lead-exposed children, including significant decreases in high school graduation. Other findings such as decreased coordination, balance, hand/eye coordination, and various abnormalities in personality measures have also been found to be associated with lead exposure. Most significant is the fact that these findings are often dose-related, with toxic levels of lead exposure associated with blood-lead levels as low as 10 µg/dL. It is these findings that resulted in the 1991 CDC directive to lower admissible childhood levels of blood-lead to no greater than 10 µg/dL (CDC, 1991). Twenty years ago, lead toxicity was considered medically significant only at the level where children experienced overt symptoms such as abdominal cramping, seizures, anemia or other obvious physical findings at blood-lead levels of approximately 50 µg/dL or greater.

Figure 2. Lowest Observed Effect Levels (µg/dL) of inorganic lead in children. [1]



(1) From ATSDR, 1990.

Current standards have resulted in screening programs, including those in the state of Michigan, which are targeting children in high risk areas, such as impoverished areas or inner cities. Identification of children with elevated blood-lead levels, especially those in the lower ranges of 10 µg/dL to 20 µg/dL, allow for removal of sources of lead exposure, such as house paint. Also, such programs facilitate prevention of subsequent exposure to siblings of the identified child, or other children visiting the home. Unfortunately, there is no medical treatment for children with blood-lead levels in the range of 10 µg/dL to 40 µg/dL. Chelation, the only known medical treatment which removes lead from the body, is reserved for children generally with levels of at least 40 µg/dL or greater. At lower levels, it is felt by many physicians that the potential adverse side effects from chelation treatment may outweigh the long-term benefits.

Special Effects in Adults

The most significant lead exposure among adults comes through occupation. Lead exposure while at work typically results from inhalation of either fumes or dust. Such exposures are most prevalent in the construction trades, but can occur in a variety of

other occupations, especially those involved in removal of lead-based paint (National Center for Lead-Safe Housing, 1994).

As with children, the effects of lead poisoning in adults are insidious. Initial presentation often includes irritability and mood swings. Early symptoms also include insomnia, headache and a metallic sweet taste in the mouth. Subsequently, symptoms such as loss of appetite, diffuse muscle pain, joint tenderness, and tremor develop. Specific organ effects are described above (Zenz, 1988).

The neurobehavioral effects of lead exposure, particularly at the lower levels of blood-lead levels ranging from 10 µg/dL to 20 µg/dL, are less of a concern for adults. The likely explanation for this is the fact that brain development is active in young children, but is generally complete by age six. However, long-term central nervous system effects in adults have received minimal study to date. Outcomes such as decreased intelligence, motivation, and personality change, for example, could adversely affect job productivity, family and other social relationships, and requires further study.

Exposure Pathways

The risk of environmental exposure to lead is greatest for children (< 6 years old) when mental development is the greatest. Nearly every reported case of elevated blood-lead in this age group is due to ingestion of lead-based paint chips or dust generated in a house with peeling lead-based paint (USEPA, 1986a). Although the overwhelming exposure is due to paint containing lead, there are numerous other exposure routes that contribute to the overall body burden and, in some cases, can cause an additional incremental burden that is sufficient to cause an adverse effect. These other exposures can be from air, water, soil and food. Each of these routes will be discussed below, and a total lead exposure assessment will be developed.

Lead-based Paint. Lead-based paint remains the most common high-dose source of lead exposure for young children. Paint containing up to 50% lead was in widespread use through the 1940's (CDC, 1991). Usage declined after that but even today, 74% of all privately owned homes in the U.S. built before 1980 contain lead-based paint (USHUD, 1990a). In 1978, the Consumer Product Safety Commission banned the manufacturing of paint containing more than 0.06% lead by weight on interior and exterior residential surfaces, toys, and furniture (CDC, 1991). Lead-based paint is still available for industrial, military and marine uses, however.

In well-maintained homes, lead exposure is not a problem. Problems occur when the paint is allowed to crack and chip. This is especially prevalent in areas such as window sills that are prone to moisture damage. As the paint flakes off it becomes part of the household dust. Children playing and crawling on the floor readily transfer the dust from the household surfaces to their mouths. In homes without lead-based paint problems, household dusts contain about 300 µg/g of lead dust (USEPA, 1986a). Dust contaminated by chipping lead-based paint can contain several thousand µg/g of lead-containing dust. In addition, pica, the repeated ingestion of nonfood substances, has been implicated in many cases of child lead poisoning. Because of its sweet taste, the

lead-based paint chips are even more appealing.

Airborne Lead. Major sources of airborne lead include combustion of leaded gasoline, industrial processes, and incineration. Prior to the phaseout of leaded gasoline, which began in 1975, U.S. lead emissions peaked at 153,100 metric tons/year in 1976 with vehicles burning leaded gasoline accounting for 136,600 metric tons per year or 89% of the total (USEPA, 1986b). On July 1, 1985, the allowable lead content of leaded gasoline was reduced from an average of 1.0 g/gallon to 0.5 g/gallon, and on January 1, 1986 to 0.1 g/gallon. Furthermore, unleaded gasoline was introduced in 1975 for vehicles equipped with catalytic converters. By 1992, unleaded gasoline accounted for 99% of gasoline sales (USEPA, 1993). The effects of this phaseout have been dramatic (see Table 6).

Table 6. Sources of airborne lead in the U.S. in 1976 and 1993 (units are in 1,000 metric tons per year). **(1)**

Source Category	1976	1993
Transportation	136.4	1.6
Fuel Combustion	8.3	0.5
Industrial Processes	8.1	2.4
Solid Waste Disposal	4.3	0.7
Total	**153.1**	**5.2**

(1) From USEPA, 1993; 1986b.

In 1978, the USEPA promulgated a National Ambient Air Quality Standard (NAAQS) for lead of 1.5 $\mu g/m^3$ averaged over a 3-month period. In 1977, the year prior to the promulgation of the NAAQS, the mean concentration of lead in ambient air in U.S. cities was about 1.3 $\mu g/m^3$ with a number of cities exceeding the NAAQS (USEPA, 1986b). In Michigan, no violations of the NAAQS occurred until 1978 when excursions were recorded in both Grand Rapids and Detroit. In Grand Rapids, the maximum concentrations reached 1.6 $\mu g/m^3$ and in Detroit, it reached 2.32 $\mu g/m^3$ (Heindorf, 1994). Both sites are heavily influenced by freeway traffic.

Ambient air concentrations followed the same trends as the emissions. The 1993 average concentrations recorded in Michigan are shown in Table 7. A comparison of the values in Table 7 to the 1978 values indicates that airborne concentrations of lead in Michigan have declined approximately two orders of magnitude since the elimination of leaded gasoline. Nationwide, the trends are similar, but there are six sites that still violate the NAAQS. In all six cases, the violations are due to the proximity of large industrial sources, generally non-ferrous smelters. In Michigan, there do not appear to be any point-source hot spots.

Water. The concentration of lead in surface waters in Michigan is generally less than 10 µg/l with a decreasing trend over the past ten years or so (Sills, 1994). In contrast, tap water samples taken from homes average about 10 µg/l to 15 µg/l and can range into the thousands of µg/l. The reason is that lead is leached out of the plumbing within the

distribution system. Sources of lead within the distribution system include, lead pipe connections from under the street to the homes, water meters, leaded connections, and soldered joints. In addition some well pumps are a source (Deininger, 1994).

Table 7. Ambient air arithmetic mean concentrations of lead in Michigan in 1993. **(1)**

City/Town	Site	Lead ($\mu g/m^3$)
Flint	Whaley Park	0.01
Kalamazoo	Fairgrounds	0.02
Grand Rapids	College	0.01
Wyoming	28th St.	0.01
Muskegon	Bennett	0.01
Lawton	880	0.01
Detroit	Evergreen	0.02
Detroit	W. Fort	0.03
Detroit	Linwood	0.03
Detroit	E. 7 Mile	0.02
Dearborn	Wyoming	0.04
Grosse Ile	W.River Rd.	0.02
Allen Park	Moran	0.03
Menominee	17th Ave.	0.03

(1) From MDNR, 1994.

Soil and Road Dust. A 1992 Michigan Department of Natural Resources (MDNR, 1992) survey of lead in soils and roadside dusts indicates that background lead levels in Michigan soils range from 2.5 µg/g to 55 µg/g. Lead in roadside dusts range from 29 µg/g to 430 µg/g with a mean concentration of 142 µg/g.

Food and Beverages. Food represents a significant route of lead ingestion. Lead occurs in and on food naturally in varying amounts and from atmospheric deposition. Lead is also added to the food as it is harvested, transported, processed, packaged, and prepared. Sources of this lead include dust, metals used in grinding, crushing, sieving, solder use in packaging, and water used in cooking. The USEPA (1986a) estimates that the average American 2-year old child, male adult, and female adult ingest 18.9 µg/day, 35.8 µg/day, and 25.3 µg/day of lead in food and another 4.1 µg/day, 10.3 µg/day, and 5.9 µg/day in beverages, respectively. It should be noted that between 1973 and 1978, intensive efforts were made by the food industry to remove sources of lead from infant food items. Most of the reduction was made by the discontinuation of soldered cans used for infant formula. Since then, the can manufacturers have discontinued making soldered cans for the food industry (USEPA, 1986a).

Baseline Estimates of Lead Exposure in Michigan Children

In its 1986 Air Quality Criteria Document for Lead, the USEPA (1986a) estimated baseline exposures associated with different sources of lead for a 2-year old child, an adult male and an adult female. Since the greatest concern here is for children, the USEPA procedure and assumptions used for the child are described below and then altered to better reflect baseline and worst case exposures which are more specific for Michigan. The sources of lead include air, food, water and beverages, and dust.

Air. The total amount of lead inhaled by a 2-year old child was estimated to be 0.5 µg/day (USEPA, 1986a). The following assumptions were made by the USEPA (1986a):

(1) the average ambient air lead concentration was 0.1 µg/m^3 and (2) the ratio of indoor to outdoor concentrations was 0.5. The mean concentration of ambient lead in Michigan in 1993 was about 0.02 µg/m^3, and the highest concentration, which was observed in Dearborn, was 0.04 µg/m^3 (MDNR, 1994). Using the Michigan ambient lead concentrations, the estimated inhalation of lead would be 0.1 µg/day on average and 0.2 µg/day for the highest concentrations (worst case).

Food. The USEPA (1986a) estimated that a typical 2-year old child ingests 18.9 µg/day of lead through food. Of that, the agency estimated that 9.2 µg/day was due to deposition of airborne lead onto the food. The airborne concentration again was assumed to be 0.1 µg/m^3, so an adjustment was needed to reflect the lower concentrations that now exist in Michigan. When these adjustment are made, the resulting mean and worst case lead consumption rates are 11.5 µg/day and 13.4 µg/day, respectively.

Water and Beverages. The USEPA (1986a) estimated that the average 2-year old ingests 3.5 µg/day of lead from liquids other than water. For water containing a mean lead concentration of 10 µg/l, the agency estimated that the ingestion rate was 3.4 µg/day. The data presented by Deininger (1994) for Michigan drinking water indicate that a mean concentration of 10 µg/l is a reasonable assumption. Therefore, the mean amount of lead ingested from water and beverages is 6.9 µg/day. Deininger's (1994) data also suggest that the worst few percent of homes have water lead levels greater than 60 µg/l, and some houses exceed 100 µg/l. Using the worse case scenario of 100 µg/l, lead ingestion from water would be 34 µg/day and the total from beverages and water would be 37.5 µg/day.

Dust. The USEPA (1986a) estimated that the average child living in a house that does not have peeling lead-based paint ingests 21 µg/day of lead in dust. This includes 15 µg/day of household dust at a concentration of 300 µg/g of household dust, 4.5 µg/day of outside dust at a concentration of 90 µg/g, and a 1.5 µg/day of industrial dust at a concentration of 150 µg/g. The 300 µg/g of lead in the household dust was based primarily on data collected before the phaseout in the use of leaded gasoline. Since the source of this dust (in the absence of a lead-based paint source) is assumed to be atmospheric deposition, the concentrations today in Michigan homes should be considerably lower. As previously indicated lead concentrations in ambient air have decreased about two orders of magnitude since 1978. Assuming that household lead levels also decreased during this time period and using a conservative estimate of 67% (to 100 µg/g) for the decrease, the level of ingested household lead-containing dust would be expected to be about 5 µg/day.

The MDNR road dust survey (MDNR, 1992) showed that the mean concentration of lead in the road dust was about 142 µ/g. This would increase the mean ingested lead-containing road dust to 7.1 µg/day. Using the USEPA's lead value for industrial dust, the sum of the average ingestion of lead-containing dust would be 13.6 µg/day.

Based on data presented by Laxen, Raab and Fulton (1987) and Vostal *et al*. (1974) from houses with pealing lead-based paints, a household lead dust concentration of 2,000 µg/g is not unreasonable under worst case conditions. This results in an ingestion rate of

100 µg/day. Using 400 µg/g, which is the 95th percentile estimate from the MDNR survey as the concentration of lead in the road dust, an ingestion rate of 20 µg/day may be obtained. Assuming the industrial dust contribution remains the same, the total lead ingested for the worse case conditions would be 121.5 µg/day.

Total Exposure. Total estimated exposure from all sources is summarized in Table 8. Also shown in Table 8 are the ingestion estimates for an ambient air lead concentration of 2.32 µg/m^3 which corresponds to the maximum observed concentration that occurred in Detroit in 1978. All other media concentrations are assumed to be the average estimates.

Table 8. Calculated ingestion estimates of lead by children under several exposure scenarios (units are µg/day).

Source	Michigan Average	Worst Case	Detroit, 1978
Air	0.1	0.2	11.6
Food	11.5	13.4	18.9
Water	3.4	34.0	3.4
Beverages	3.5	3.5	3.5
Household Dust	5.0	100.0	15.0
Other Dusts	8.6	20.0	8.6
Total	**32.1**	**172.6**	**61.0**

Table 8 illustrates several important points. First, it does not include pica. Clearly, the ingestion of a few chips of lead-based paint could overwhelm the exposure from any other source. Second, the contribution from airborne lead is no longer important in Michigan. This was not the case in 1978, however. Household dust and water have the potential to be the two most important sources in the absence of pica.

Relationship Between Ingestion and Blood-Lead Levels in Children

The relationship between the amount of ingested lead and blood-lead level is a complex relationship and will likely vary among individuals. This is clearly demonstrated in a 1982 study of infants by the United Kingdom Central Directorate on Environmental Pollution (as cited in USEPA, 1986a). With this caveat in mind, the following analysis should be viewed with caution. The results are certainly not robust but, nevertheless, they are useful in placing ingestion rates in perspective. The basis for the analysis is the Ryu *et al*. (1983) study described by USEPA (1986a). A blood-lead level to lead ingested relationship is developed for 25 formula-fed infants from eight to 196 days of age. Up to day 112, the infants ingested an average of 17 µg/day of lead and their mean blood-lead level was 6.1 µg/dL. After day 112, the infants were separated into two groups. One remained on the formula and had an average of ingested lead of 16 µg/day. By the end of the study on day 196, this group had an average blood-lead level of 7.2 µg/dL. The second group of babies was place on whole cow milk on day 112 and their lead ingestion averaged 61 µg/day. By the end of the study, this group had an average blood-lead level of 14.4 µg/dL. The 7.2 µg/dL increase in blood-lead level per 45 µg/day increase in ingested lead corresponds to a slope of 0.16. Assuming a linear relationship between blood-lead and ingested lead, Ryu *et al*. (1983) derived the following equation:

Blood-lead = *A* + (0.16) (Ingested Lead)

where *A* would represent some background blood-lead level. Substituting 14.4 µg/dL for blood-lead and 61 µg/day for ingested lead in the equation and solving for "A", a value of 4.6 is obtained. Using the calculated value and the lead exposure estimates from Table 8, estimates for three Michigan scenarios may be made (see Table 9). Although the estimated blood-lead levels presented in Table 9 are highly uncertain, they do help to put the estimated ingested lead levels into perspective.

Table 9. Estimates of blood-lead levels in infants for given levels of ingested lead.

Ingested Lead (µg/day)	Estimated Blood-Lead (µg/dL)
32.1	9.7
172.6	32.2
61.0	14.4

Discussion and Conclusions

Sources of lead are pervasive throughout society, and perhaps even more so in Michigan because of the industrial nature of the state. Lead exposure prevention is critical to Michigan, because lead poisoning is a preventable condition, and because the insidious and subclinical outcomes of lead exposure may, in the long term, result in significant decreases in overall health and productivity of the state's citizens.

There is consensus within the scientific and medical communities that the primary routes of lead exposure in children are ingestion of lead-based paints and lead-containing dusts and soil (National Center for Lead-Safe Housing, 1994; NCEMCH, 1994; Clements International Corporation, 1993; CDC, 1991), and, in adults, inhalation of lead-containing dusts and fumes (Clements International Corporation, 1993; USEPA, 1986a). Many lead poisoned children are asymptomatic (Bhambhani, 1994). The impacts of elevated blood-lead levels in children may result in deficits in cognitive functions, neurophysiological difficulties and problems in school (Pounds, 1994; Needleman *et al*. 1990).

The elimination of the use of lead-based gasoline has measurably reduced the risk of elevated blood-lead levels to the general population. However, a calculated exposure level (31.1 µg/day) estimated to be the average currently in Michigan children roughly corresponds to the CDC (1991) blood-lead level of concern (10 µg/dL). As a consequence, there would appear to be little or no margin of safety for any additional incremental exposure in a household from peeling and deteriorating lead-based paint or lead-contaminated plumbing systems. In addition, any incidence of pica would result in a blood-lead level in excess of 10 µg/dL. The results of this analysis lends support to the observation by Bhambhani (1994) that all children in Detroit are at risk of elevated blood-lead levels. As a consequence of the inadequate and incomplete blood-lead level data base for children, this observation may be relevant to other areas in Michigan as well. It should be noted that recent historical exposures (pre-1980's) were likely higher before

airborne lead was eliminated as a source of lead exposure.

Screening is essential in order to identify children with elevated blood-lead levels. Accurate and complete data regarding exposure and blood-lead levels in populations at risk are essential in order to design and implement an effective intervention and control strategy. Comprehensive blood-lead data are not available for either Michigan children or adults. Limited blood-lead data available on lead exposure among Michigan children from three pilot study areas (city of Detroit; Ingham, Kent, Muskegon and Saginaw Counties; and Wayne County) appear to be under-reported, fragmented and incomplete. In addition, the collection, reporting and management of these data among the pilot areas are not uniform. This may be due in part to the fact that the three Michigan pilot programs are responsible to different federal funding agencies (CDC and USHUD) with different reporting requirements or expectations. The state's adult lead program, partly because of the lack of promulgated rules and mandatory reporting, has an even more serious data collection problem.

Given the above, the MESB Panel concludes that: (1) there is a need within the state to uniformly establish the use of a venous/direct lead, rather than a capillary/EP, blood-lead sampling/testing protocol, and to use 10 µg/dL, rather than 15 µg/dL or 20 µg/dL, as the blood-lead level reporting statistic for children across the state; (2) there is a need for the state to take the lead in terms of all federally-funded funding projects dealing with lead exposure, prevention and abatement; (3) there is a need to establish a statewide, uniform and coordinated data collection, reporting and management system to capture and accurately portray the incidence of elevated blood-lead levels in both children and adults; (4) there is a need for the state to establish a lead registry for both adults and children; and (5) there is a need for the state to promulgate rules which will help enforce reporting of elevated blood-lead levels for both adults and children.

DIRECTIVE 2. Prioritize the most effective targets for remediation in terms of human exposure reduction, paying special attention to reducing exposure to children.

Introduction

Comprehensive data on the extent and level of human, and in particular childhood, lead exposure in Michigan are not available. However, based on the literature and the limited blood-lead levels and demographic data presented in Directive 1 for the state, the largest population group and also the one most at risk for elevated blood-lead levels within Michigan would be the lower socio-economic urban preschool children (< 6 years old) living in older (pre 1980's) homes. Subject to confirmation through development of accurate and comprehensive blood-lead data for the state, available state resources should be directed towards a program which would reduce lead exposure in this population. It would be envisioned that such a program would entail four essential elements; (1) Development of a lead-exposure comprehensive data base, (2) Identification and prioritization of lead-based paint problem areas, (3) Abatement of the critical lead-based paint problems and (4) Education of the resident families on home maintenance and nutrition.

Target Population Data Collection

An essential first step to any program aimed at reducing lead exposure among Michigan urban children is the development and implementation of an accurate and comprehensive database for information collected on demographic, housing and blood-lead levels information. A second step is development of a program which will ensure the consistent and coordinated collection of the data to be stored on a state-wide basis. Recommendations for improved collection and coordination of blood-lead level and demographic data have been provided in Directive 1. Through a U.S. Department of Housing and Urban Development (USHUD) grant, the state will be creating a database to store geographic and other information on available lead safe housing (MDPH, 1993). The database, referred to as the "Statewide Lead Safe Housing Reference" will be available to the public through the "IMAGIN" network; a computerized database located within the public library system.

Baltimore Jobs and Energy Project

One program which appears to be successful and incorporates both abatement and education is the Baltimore, Maryland Jobs and Energy Project. At the October 4, 1994 MESB Lead Panel meeting, Mr. Dennis Livingston outlined the community-based lead hazard reduction program currently underway in Baltimore, Maryland (Harrison, 1994a). According to Livingston (1994) the majority of the lead paint problems are found in single family units. The Baltimore Jobs and Energy Project was designed to provide affordable lead-paint and dust hazard identification, remediation and prevention program for single family homes, duplexes and small apartment buildings. The Baltimore approach is in contrast to the USHUD program which is designed to remediate lead-based paint problems in large USHUD multi-family housing projects.

The basic components of the Baltimore lead hazard reduction program include an identification and evaluation of the extent of the lead problem on a community, neighborhood, or apartment complex basis and an assignment of an abatement schedule based on the above evaluation and a needs assessment. The needs assessment includes, but is not limited to, the number of vulnerable children present, level of lead dust on surfaces, level of lead paint decay, size of surfaces to be treated, rate of lead dust generation, structural dust retention capabilities, and blood-lead levels of the resident children. The actual lead abatement work is conducted by small local contractors and local volunteers appropriately trained to the level of work in which they would be involved. Abatement is followed by education of the residents on proper maintenance of the abated or partially abated home (Livingston, 1994).

According to Livingston (1994), the Baltimore program has been successful because it has been affordable; it selectively addresses areas in need of lead-based paint and dust abatement rather than arbitrarily abating all lead-based painted surfaces; it meets USHUD clearance standards (a safe environment is achieved); and the local contractors and volunteers work in a safe environment. The key element to the success of the program is the establishment and maintenance of community-based volunteer hazard abatement crews.

Discussion and Conclusions

The development of a community-based urban lead hazard reduction program similar to that which is currently operating in Baltimore coupled with a program to ensure collection of valid blood-lead levels, demographic and housing stock data should help to reduce lead exposure among urban Michigan preschool children and should be considered by the state. Presented below is a brief outline of suggested components to be included in such a lead abatement strategy. These components, although not inclusive and containing elements of a USHUD assistance grant to the state, are viewed by the MESB Lead Panel as important elements to the success of an effective urban lead hazard reduction strategy.

1. Development of a comprehensive lead-exposure database which would:

A. Increase the number of childhood blood-lead level screenings and assure that all screenings use a venous/direct lead sampling/testing protocol (see discussion under Directive 1),

B. Improve collection, coordination and management of blood-lead level data among state and local agencies and the medical community (see discussion under Directive 1), and

C. Create a comprehensive information system which would assist government policy makers, MDPH and Michigan citizens to:

(1) Identify lead safe housing in Michigan urban areas, and

(2) Track changing trends in blood-lead levels among both children and adults.

2. Identification and prioritization of the extent of the existing lead-based paint and dust problem within the urban areas.

A. Evaluation of the extent of the lead-based paint problem within a community, neighborhood or apartment complex and development an abatement schedule based upon the problems identified.

B. Development of a needs survey which would include the following considerations:

(1) Number of vulnerable children in each household,
(2) Degree of lead-based paint decay in households with vulnerable children,
(3) Number of lead-based paint dust-collecting surfaces in households,
(4) Size of surfaces to be treated, and
(5) Rate of lead-based paint dust generation.

C. Initiation of blood-lead level testing and evaluation by local health departments of vulnerable children in homes identified with lead-based paint and dust hazards in the needs survey.

3. Abatement of needed lead-based paint and dust problems (see discussion in Directive 3).

A. Training of contractors and resident volunteers on lead-based paint and dust hazard identification, remediation, and maintenance procedures.

B. Temporary relocation of the resident family during the period of abatement (approximately one to two days), and

C. Abatement of identified lead-based paint and dust hazards during the period of family relocation by a local contractor and trained community volunteers.

4. Education of the resident family on proper home maintenance to reduce future lead-based paint and dust hazards and on proper nutrition for vulnerable children (see discussion under Directive 3).

In addition to the above listed components, and based on the Baltimore experience (Livingston, 1994), there are several legal, economic and policy issues which may arise with the development of a community-based lead hazard reduction program. Some of these issues could include: identifying and maintaining a sufficient number of resident volunteer workers; level and training certification for resident volunteers for abatement work; conflicting landlord and tenant rights, responsibilities and liabilities; contractor

liability; and cost constraints on property owners. The most effective manner to address and resolve these issues is beyond the charge and scope of this evaluation. State policy makers and regulators will nonetheless need to be cognizant of these possible constraints.

Finally, and given the magnitude of instituting a statewide urban lead hazard reduction program, the MESB Lead Panel would suggest that prior to development of a such a program that the state: (1) search out other similar programs currently operating within the country; (2) further investigate, and possibly actually visit, the Baltimore project to become familiar with both the successes and failures of the program; and (3) pilot a lead hazard reduction program in one or two selected urban areas.

DIRECTIVE 3: Identify the efficacy of various remediation techniques for lead.

Introduction

The purpose of this portion of the report is to identify the efficacy or effectiveness of the various remediation (abatement) strategies to mitigate human exposure to lead from paint, dust, soil and drinking water. To date, there have been several investigations designed to determine the effectiveness of lead-based paint abatement measures (Buxton *et al*., 1994; Staes *et al.*, 1994; Amitai *et al*., 1991; USHUD, 1991; Farfel and Chisolm, 1990a; 1990b; soil and dust (Weitzman *et al*., 1993; Charney *et al*., 1983); and drinking water (Cosgrove *et al*., 1989; Moore, Richards and Sherlock, 1985; Karalekas, Ryan and Taylor, 1983). In addition, there are several ongoing investigations designed to look at the efficacy of lead-dust cleaning, lead-paint repair and maintenance, encapsulation protocols, and lead cleaning products (Cook, 1994).

Lead-based Paint, Soil and Dust Abatement

Under the federal Residential Lead-Based Paint Hazard Reduction Act of 1992 and the draft U.S. Department of USHUD Guidelines for the Evaluation and Control of Lead-based Paint Hazards in Housing (National Center for Lead-Safe Housing, 1994) framework, lead-based paint hazard controls are categorized into three types: interim controls, abatement of lead-based paint hazards and complete abatement of all lead-based paint. Interim controls are designed to address hazards quickly, while both abatement controls are intended to produce a more permanent (at least 20 years) solution.

Interim controls are a set of measures designed to reduce temporarily human exposure or likely exposure to lead-based paint hazards. Such controls may include dust removal, paint film stabilization, and treatment of friction and impact surfaces. Education, ongoing maintenance and monitoring, and periodic reevaluations by certified professionals are also a part of interim controls. Interim controls for lead-contaminated soils include covering with grass or gravel, or land-use controls, such as fences, bushes or decks. The interim controls are designed to temporarily reduce exposure. How long the soil interim controls remain effective depends on many factors, including the durability and maintenance of the cover, degree of foot traffic and climate.

Abatement of lead-based paint hazards means the removal of lead-based paint and lead-contaminated dust, the permanent containment or encapsulation of lead-based paint, the replacement of lead painted surfaces or fixtures and the removal or covering of lead-contaminated soil. Encapsulation includes coatings and rigid coverings that are bonded to the existing paint film with an adhesive. Encapsulation is different from a permanent enclosure system which is mechanically fastened to the structural system and not dependent on the substrate coating for durability.

Complete abatement means the permanent elimination of all lead-based paint, interior or exterior, intact or not intact. Complete abatement would include permanent enclosure systems. Abatement of lead-contaminated soils would include the complete removal of at

least the top six inches of soil, soil cultivation, soil treatment and replacement, or paving with concrete or asphalt.

The efficacy of a given form of lead hazard control (interim control or abatement) may be measured by how well it eliminates or at least reduces an individual's environmental exposure to lead-contaminated materials and how well its implementation reduces the blood-lead levels of exposed individuals. Burgoon, Rust and Schultz (1994) recently reviewed the relevant available scientific literature regarding lead-based paint, dust and soil interim controls and abatement. They identified 14 studies which were conducted during the period 1974 to 1993 where one or more lead-based paint, dust or soil interim control or abatement technique was used. Of the 14 studies identified, only 10 also evaluated the change in blood-lead levels in conjunction with the control/abatement technique. All 14 investigations encountered some degree of confounding problems either in the initial design or as a result of some unexpected occurrence during the study. In addition, none of the studies involving blood-lead level comparisons controlled for the possibility of mobilization of accumulated lead in the affected residents' bones following an intervention, or the impact of seasonal variation in blood-lead and environmental lead levels. Both factors may moderate or magnify the perceived effectiveness of the particular intervention.

Burgoon, Rust and Schultz (1994) summarized the results of their review by indicating that:

1. The evidence from the nine lead-paint abatement investigations (Battelle Memorial Institute and Midwest Research Institute, 1994; Buxton *et al.*, 1994; Staes *et al*., 1994; Markowitz *et al.*, 1993; Ruff *et al*., 1993; Amitai *et al*., 1991; Rosen *et al*., 1991; USHUD, 1991; Farfel and Chisolm, 1990a; 1990b; and Copley, 1983) suggested that the efficacy of these methods was dependent, in part, upon the safeguards employed to protect the occupants and their residential environment during abatement;

2. Average blood-lead levels in two lead-based paint abatement studies (Amitai *et al*., 1991; Farfel and Chisolm, 1990b) were observed to increase 16% to 19%, on average, during abatement and remained elevated following the intervention. In both studies, elevated blood-lead levels were associated particularly with dry scraping paint removal methods;

3. Lead-based paint encapsulation, enclosure and replacement methods in one study (Amitai *et al*., 1991) were associated with an average blood-lead reduction of 2 µg/dL to 3 µg/dL;

4. Lead-based paint removal methods (enclosure, removal) were shown to lower the blood-lead levels of participants in several studies (Staes *et al*., 1994; Markowitz *et al*., 1993; Ruff *et al*., 1993; Amitai *et al*., 1991; Rosen *et al*., 1991; and Copley, 1983) by 20% to 29%;

5. Evidence that incomplete lead-based paint abatement (enclosure, encapsulation and removal) may not be fully effective (as measured by lead-containing dust levels in abated

versus unabated homes) was found in one study (Buxton *et al*., 1994; Battelle Memorial Institute and Midwest Research Institute, 1994). As was expected, certain unabated areas within the homes (e.g., air ducts) and outside the homes (e.g., soils) showed higher lead-containing dust loadings in abated versus unabated homes. Also, higher but not significant mean lead-containing dust loadings were found in abated versus unabated homes;

6. Soil-lead and floor dust-lead levels, where source isolation or removal and in-place management methods (soil removal, stabilization of peeling paint and wet mopping of interior dust) were used in abated residences, remained low following intervention (Weitzman *et al*., 1993). The blood-lead levels of affected residents oscillated but did not return to pre-intervention levels. After one year blood-lead levels were found to have declined, on average, by 1 µg/dL to 2 µg/dL; and

7. In-place dust management methods (e.g., post-abatement wet mopping) do not appear to aggravate childhood lead exposure if performed properly (Charney *et al*., 1983). Once the dust technique was eliminated, the lead-containing dust hazard returned (Charney *et al*., 1983; Weitzman *et al*., 1993). Regular, extensive dust hazard management efforts by trained personnel produced an 18% decline in mean blood-lead concentration in affected residents (Copley, 1983).

Burgoon, Rust and Schultz (1994) concluded that the 14 studies suggested that both in-place management (interim control per USHUD definition) and source isolation or removal (abatement per USHUD definition) techniques for lead-based paint, and lead-laden soil and dust were at least partially effective in reducing blood-lead concentrations. Source isolation or removal methods often had an accompanying risk of at least short-term elevation of residents' blood-lead levels. In-place management methods, in turn, usually required sustained efforts to retain their effectiveness. Overall, there was no definitive evidence in the literature that suggested that either of these methods categories was more efficacious than the other.

A recent study (Swindell *et al.*, 1994) on pre- and post-abatement blood-lead levels of children from lead-abated homes suggests that current abatement procedures may be limited in terms their effectiveness in reducing blood-lead levels. Swindell *et al*. (1994) studied the effect of home abatement on blood-lead levels in 132 children who had not undergone chelation therapy and whose homes were abated between 1987 and 1990, when strict abatement (and clean up) guidelines were enacted. The results of the study indicated that in the majority of children with blood-lead levels equal to or greater than 25 µg/dL, and particularly above 30 µg/dL, residential de-leading was associated with an 18% decrease in venous blood-lead levels in the year following abatement. However, when the child's pre-abatement blood-lead level was below 25 µg/dL, and particularly below 20 µg/dL, the child's blood-lead level was more likely to increase than decrease following abatement. Swindell *et al.* (1994) concluded that if home abatement is to be effective for children with blood-lead levels below 30.0 µg/dL, and particularly below 20.0 µg/dL, improved abatement standards and practices will need to be devised, tested and implemented.

Drinking Water Lead Abatement

According to the USEPA (1986a), about 20% of the population is exposed to elevated lead levels above 20 µg/L in first-draw tap water. Lead occurs in drinking water primarily due to corrosion of lead-bearing materials in water supply distribution systems (e.g., service lines, goosenecks, and water meters) and in household plumbing (e.g., lead-soldered copper pipes, brass faucets, and brass fixtures). The 1986 Safe Drinking Water Amendments banned the use of lead solder from public water supply systems and from residential or non-residential facilities connected to a public water system. The use of pipes or faucets containing more than eight percent lead was also banned (USEPA, 1989). The highest lead levels are found in areas with corrosive waters, especially in older urban areas with lead service lines and mains and in homes with newly-installed brass fixtures (MTC, 1993; USEPA, 1991b). Within Michigan, Deininger (1994) has indicated that the major determinants of lead concentration in home water supplies were the water meter, in-house plumbing and the faucet rather than the service line. Both zinc and brass components of faucets and water meters contain lead impurities that leach into stagnant water. Within rural homes the plumbing components of concern would be submersible pumps with brass parts, in-house plumbing and faucets (Deininger, 1994). According to Lee, Becker and Collins (1989), brass fixtures may account for approximately one-third of the lead in the first-draw water.

Lead in plumbing systems may be eliminated by replacing lead pipes and/or removing and replacing solder and fluxes with tin/antimony or silver/tin solders as soldering material for plumbing in drinking water systems. Another approach to prevent leaching of lead in plumbing systems is to implement a corrosion control measure by pH adjustments. Two important and probably more practical measures are to: (1) flush the stagnant water from the system prior to its use for drinking or cooking purposes to avoid exposure to any overnight or extended period accumulation of lead in the system (CDC, 1991; MDPH, 1991; Chadzynski, 1986) and (2) only draw fully flushed water from the cold water tap (CDC, 1991). The length of time needed to fully flush any particular system will vary from home to home depending on the lengths of pipes both inside and outside the house (Deininger and Bernstam, 1993).

Finally, questions remain regarding the use of many point-of-use devices (treatment devices installed at the tap) in reducing lead in water, since their effectiveness will vary and may be affected by the location of the device in relation to the lead source and by compliance with the manufacturer's use and maintenance instructions. Some devices, like reverse osmosis and distillation units, may be effective in removing lead. Carbon, sand and cartridge filters do not remove lead, however (CDC, 1991).

Nutritional Intervention

The mechanism and rate of lead absorption from the gastrointestinal tract is not completely understood, but it is believed that absorption occurs in the small intestines by both active and passive transport following solubilization of lead salts by gastric acids (Haddad and Winchester, 1990; Ellenhorn and Barceloux, 1988). Lead is poorly absorbed from the gastrointestinal tract; however, toxic effects can result from the

relatively small amount of lead that is absorbed. It has been estimated that approximately 10% to 20% of an administered dose is absorbed by adults and up to 50% of ingested lead is absorbed by children (Behrman *et al*., 1992; CDC, 1991).

The role of nutrition as an intervention measure in helping to reduce the impact of lead exposure during and following lead abatement programs is one which has only recently received attention. According to Bhambhani (1994), NCEMCH (1994), Clements International Corporation (1993), CDC (1991), Mahaffey (1990), and Mahaffey, Gartside and Glueck (1986), a number of nutritional factors suppress lead absorption and toxicity. In particular, studies have shown that both calcium and iron nutritional status in young children is inversely related to the lead absorption level (Clements International Corporation, 1993). According to Mahaffey (1990), rats that consumed a low calcium diet and were exposed to lead had about four-times higher blood-lead levels than did rats on a normal calcium intake, even though the amounts of lead consumed were equal. Soft tissue lead concentrations were highly elevated with low calcium diets. This effect of low calcium intake on potentiating lead toxicity was found for several other species. Iron and zinc deficiency also increased the susceptibility of rats to lead intoxication. Iron-deficient rats given lead had increased lead levels in kidney and bone compared to rats consuming equal amounts of lead and adequate amounts of iron. As dietary zinc content increases in rats, tissue-lead levels and lead toxicity decrease. Increased maternal zinc dietary intake can also reduce the transfer of lead from mother to fetus in rats. Based on these and other observations, Mahaffey (1990) has suggested that improving the nutritional status of children with high risk of lead exposure/toxicity can increase the effectiveness of environmental lead abatement, and the National Center for Education in Maternal and Child Health (NCEMCH, 1994) routinely recommends for children a sound nutritional guideline composed of regular meals rich in iron, calcium, protein, and vitamins, but low in fat, saturated fat and cholesterol as compatible with lead poisoning prevention.

Educational Intervention

One of the in-place management dust control programs reviewed by Burgoon, Rust and Schultz (1994) is of special interest since it is the only one to attempt to evaluate the impact of educational intervention on blood-lead reduction. The study (Kimbrough, 1992) was conducted in 1991 in Granite City, Illinois. Although including children ranging in age from six months to greater than 15 years, the study focused on children less than six years of age and their families. Venous blood samples, soil samples, dust samples from within the residences, tap water samples, and an assessment of the lead content in interior paint were collected. Families of children whose blood-lead levels were found to be greater than 10 µg/dL were provided extensive counseling on how to identify lead-based paint hazards, how to perform house cleaning procedures (for dust) and hygienic procedures for young children and on lead exposure prevention in general. The affected children were re-sampled for blood-lead concentration after four months. Blood-lead levels were found to have significantly lowered in all age groups of children (6 - 71 months, mean decrease = 7.2 µg/dL; 6 - 14 years, mean decrease = 5.9 µg/dL; ≥15 years, mean decrease = 7.0 µg/dL). Unfortunately, no control group was established for the study. As a consequence, it is not possible to fully determine the implications of this study.

Discussion and Conclusions

According to the National Center for Lead-Safe Housing (1994), the belief that in order to be poisoned children must eat lead paint chips is unfounded. The most common cause of poisoning in children is ingestion through hand-to-mouth transmission of lead-contaminated surface dust (Clark *et al*., 1991; Bellinger *et al*., 1991). Lead-contaminated dust is generated as lead-based paint deteriorates over time, is damaged by moisture, abraded on friction and impact surfaces, or disturbed in the course of renovation, repair or abatement projects.

The Panel concludes that while comprising a potential future hazard, intact and well-maintained lead-based paint should not be abated. This conclusion is in keeping with the USHUD 1994 draft lead-based paint guidelines (National Center for Lead-Safe Housing, 1994) which calls for greater focus on correcting lead-based paint hazards rather than abating all lead-based paint. Lead-based painted surfaces become a hazard only when they have been allowed to deteriorate. One of the main reasons, other than lack of maintenance, for lead-based paint deterioration is trapped moisture (Livingston, 1994; National Center for Lead-Safe Housing, 1994). Livingston (1994) suggests that as a consequence of most homes becoming energy efficient, moisture is more easily trapped and undermines most painted services. Homeowners with currently intact lead-based paint and contractors who abate homes with deteriorating lead-based paint should be made aware of the problem and methods to correct it.

Given the problems with the lead-abatement studies reviewed, the MESB Panel could not determine which of the specific in-place management and abatement techniques was the most effective. Future paint, soil, and dust lead-abatement studies are needed which will be well designed and controlled, and which will incorporate blood-lead measurements. However, based on the available lead-based paint, dust and soil hazard control techniques data, and on the growing evidence linking exposure to lead-containing dust to elevated blood-lead levels in children, the MESB Lead Panel does conclude that the most efficacious lead-based paint abatement protocol should include those methods which employ the least invasive, least dust generating in-place management and abatement technique(s) available.

Finally, the MESB Lead Panel concludes that all lead-based paint in-place management and/or abatement interventions should be followed with education for the adult occupants regarding identification and management of lead-based paint hazards, periodic and proper house cleaning and maintenance procedures, and proper nutrition and hygiene for children living in the home. This may be of particular importance for children with blood-lead levels below 30 µg/dL given the possible negative impact of current lead abatement techniques on lower blood-lead levels. Additional controlled studies are also needed to better evaluate the overall impacts of both education and sound nutrition on lead-abatement effectiveness.

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APPENDIX I

February 25, 1994 Letter from Governor John Engler To the Michigan Environmental Science Board

February 25, 1994

Dr. Lawrence Fischer, Chair
Michigan Environmental Science Board
Lewis Cass Building, 2nd Floor
P.O. Box 30026
Lansing, Michigan 48909

Dear Dr. Fischer:

The state of knowledge concerning lead and its impacts has been evolving rapidly. Lead has public and environmental health significance and has an enhanced exposure potential due to human activities, especially inpopulatin centers. Protecting the public from excess lead exposure is a major public health focus within Michigan today. In addition, a scientific consideration of lead risks would further address the concern about trace metals in the ecosystem cited in Michigan's report on relative risk. As a consequence, I am requesting that the Michigan Environmental Science Board thoroughly investigate these concerns about lead in Michigan in order to provide guidance to policy-makers.

Specifically, I would like the board to do the following:

1.Identify and rank the various routes of lead exposure in terms of human exposure.
2.Prioritize the most effective targets for remediation in terms of human exposure reduction, paying special attention to reducing exposure to children.
3.Identify the efficacy of various remediation techniques for lead.

I am directing the Departments of Public Health and Natural Resources to fully cooperate with and support the Board's investigation. I would encourage the Board to also seek assistance in this assignment from federal and municipal governments, the academic and scientific communities and environmental communities.

I would appreciate you providing me with your evaluation and recommendation by June 30, 1994. Thank you for your continuing service to the citizens of Michigan.

Sincerely,

John Engler
Governor